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EXCEPTIONAL TYPES OF SLOW HEART ACTION

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With Plates 14 and 15

Case I. G. K., a man aged 62, was admitted to the City of London Hospital for Diseases of the Chest on March 9, 1912, complaining of shortness of breath and cough, which had troubled him at times during the previous three or four years.

Family history. His father and mother were both dead. His three brothers were dead: one died of rheumatic fever, one of 'heart disease', and one of 'congestion of the lungs'. One sister was alive and well. The patient was married and had ten children alive and well.

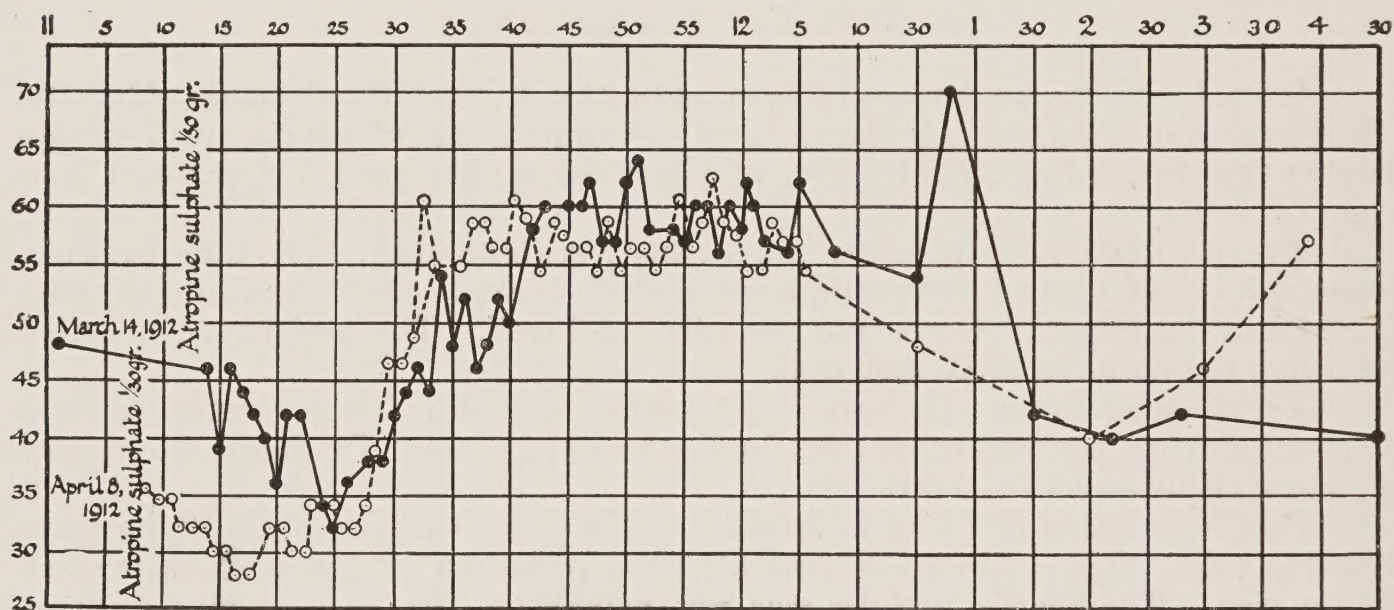
Previous illnesses. He had measles when a child. He had suffered from a cough during the winter months for the last six years. There was no history of acute rheumatism or chorea.

Condition on admission to hospital. The patient was a well-built, healthy-looking man. There was considerable visible pulsation in the neck with each heart-beat. The heart's impulse was not palpable. The deep cardiac dullness extended $1\frac{1}{4}$ and $5\frac{1}{4}$ inches to the right and left of the mid-sternal line respectively. A slight systolic bruit was present at the apex. The sounds over the other valve areas were faint. The heart was slow (40 per minute) and somewhat irregular in action. The radial pulses were equal. There was considerable thickening of the radial arteries. The liver was not enlarged. There were numerous rhonchi over both lungs, and they were conspicuous at the bases, where some crepitations were also heard. There was neither ascites nor oedema of the ankles, but the patient stated that he had noticed swelling of the left leg occasionally. The systolic blood-pressure (Riva-Rocci) was 168 mm. Hg.

Progress. The patient continued in the condition in which he was admitted for several days, and the pulse-rate remaining slow, on March 14, $\frac{1}{50}$ grain of atropin sulphate was injected subcutaneously at 11.13 a.m., and continuous polygraph tracings were taken for fifty-six minutes. The pulse-rates were calculated from strips of curve of half-minute duration in the majority of readings, the pulse-rate being originally 48 per minute (see Chart). At first a slight retardation of rate occurred. This was followed about twenty minutes after the injection by an increase of rate. The heart-rate became accelerated to 64 beats per minute, but did not go beyond this limit; the pulse became regular. By 1.30 p.m. the pulse-rate had returned to its previous slow rate. Subsequently the pulse-rate remained slow. On April 8 a subcutaneous injection of $\frac{1}{30}$ grain of atropin sulphate was administered, and a continuous tracing was taken for fifty-eight minutes following this injection, and the pulse-rates per minute calculated as before. On this occasion also a slight retardation of rate occurred within the first ten minutes of injection, and this was followed about twenty-two

¹ Working under the tenure of a Beit Memorial Research Fellowship.

minutes after the injection by an acceleration of heart-rate. At the same time the patient complained of a dry feeling in the throat. The greatest acceleration was to 62 beats per minute, and within two hours of the injection it had almost returned to its original slow rate (48 per minute). The heart maintained this low speed of contraction during the three months he was in hospital. The evening rate was usually somewhat faster than that of the morning. On May 2 he was allowed to walk down four flights of stairs, spend a half-hour in the grounds, and return to the wards. On this evening the pulse-rate was 56 per minute, and each day it was observed that similar exertion produced the same effect. The fastest rate thus observed was 64 per minute, except on a solitary occasion (May 16), when a rate of 80 per minute was attained. The general physical signs remained unaltered, but the exercise produced a certain amount of dyspnoea. No drug of the digitalis group was given at any time during his stay in hospital.



A chart constructed from continuous polygraphic curves taken from Case I. It shows the effect of two atropin injections, given on March 14 and April 8 respectively. Up till 12.10 the time scale is one division to the minute, afterwards it is two divisions to fifteen minutes.

To sum up, while the usual pulse-rate was 40–48 per minute, after a period of three months, the period of observation, it not infrequently fell below these rates: the lowest rate reached was 26. Rates of over 48 were only observed after atropin injection and exercise.

For the clinical notes of this case and the observations upon the action of atropin I am indebted to Dr. Agassiz, resident medical officer at the City of London Hospital.

Graphic records. In most of the polygraphic curves taken from this patient each arterial pulse-beat was accompanied by a venous wave of the plateau form, the latter being regularly preceded by a single *a* wave (Fig. 1). The corresponding electro-cardiographic curves showed a normal sequence and normal P summits. The slow heart action was therefore usually of uncomplicated sinus origin (Fig. 11). The slow rhythm, as has been stated, was usually fairly regular, but from time to time irregularity occurred. Under these circumstances the irregularity, as portrayed by the venous and electro-cardiographic curves, was also of sinus origin (Figs. 1 and 11); the irregularity was parallel in auricle and ventricle; each ventricular summit, wherever placed, was preceded by an auricular summit of constant form. Here and there in the polygraphic curves the

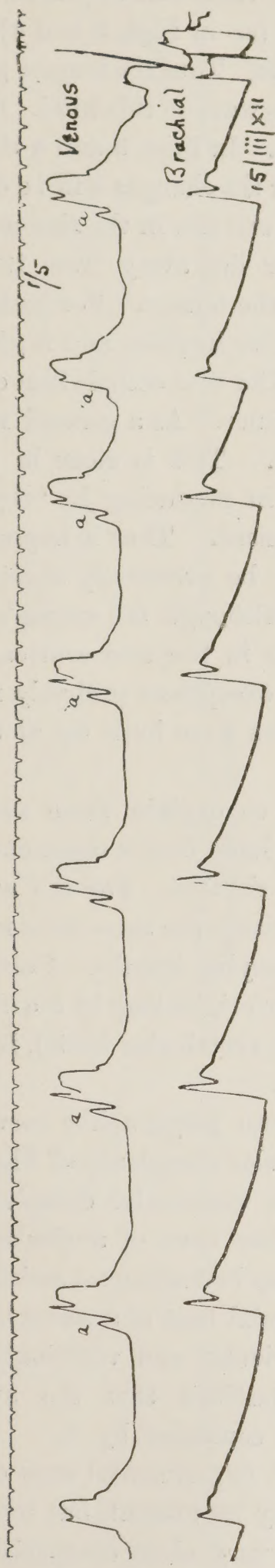


FIG. 1. A polygraphic curve from the jugular vein and the brachial artery, showing a slow and irregular action of the whole heart. The breathing was held.

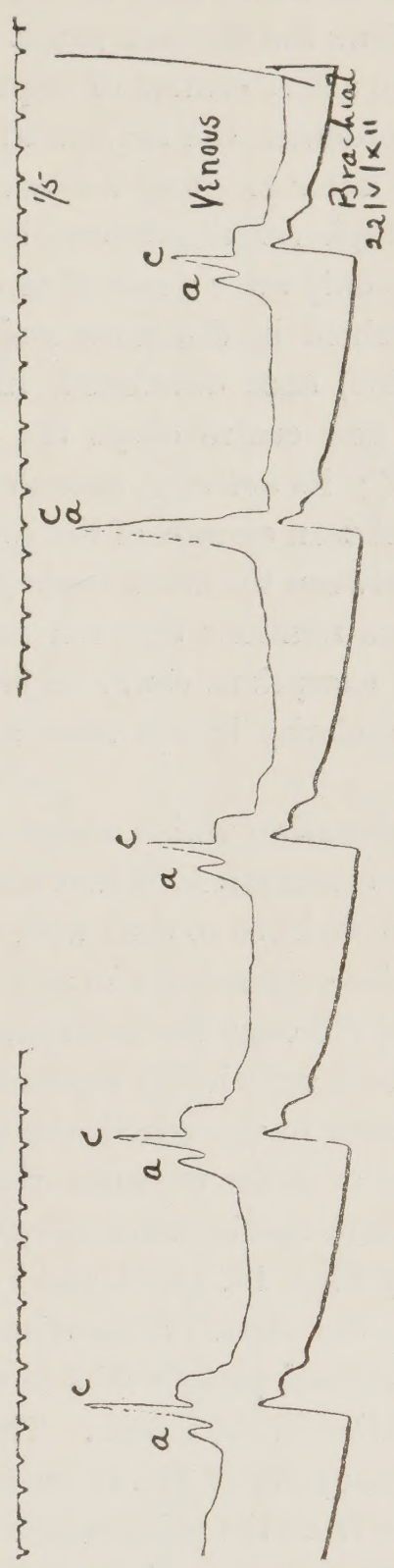


FIG. 2. A polygraphic curve from the same case as Fig. 1, showing four beats of normal origin and a beat of nodal origin, the latter occurring after the longest pause.

mechanism was seen to alter abruptly, and large venous waves, evidently the result of simultaneous contraction of auricle and ventricle, appeared. These might be isolated (as in Fig. 2) or in short groups (as in Figs. 3 and 4). As a general rule they occurred after relatively long pauses. Thus the longest pause in Fig. 2 is the only pause which is followed by a venous wave of this kind. In Fig. 4 the large venous waves start after the longest pause. In Figs. 3 and 4 the usual sequence follows the shortest pause. The reason for the changes will be obvious. There are two active centres of impulse-formation; one lies in the sino-auricular region and produces the sequential beats, the other lies away from it and is ectopic; the latter produces a slower rhythm than the former. For instance, in Fig. 2 the ectopic centre produces but a single effective impulse, and it gives rise to a response only after a rest of unusual length. The first ectopic beat of Fig. 4 may be explained in the same and customary manner. As a general rule, the ectopic rhythm, once developed, increased in rate. This is clear in Figs. 3 and 4. The new centre obeys the customary law in producing its 'rhythm of development'; its activity, once awakened, is enhanced. Thus it happens that the pauses between certain of the ectopic beats may be eventually shorter than the pauses between the sinus beats (as in Fig. 3). Although the examples given illustrate these relations between impulse-formation in the two centres, yet in a few curves exceptions could be found, and these exceptions probably resulted from an irregularity in the rates at which impulses were built up at the two points.

The explanation which alone seems adequate to explain these successive simultaneous contractions is that they have been let loose from a common centre; in short, that we have to deal with transient nodal rhythm. For the auricular and ventricular contractions in any cycle have precisely the same time relations to each other, although the individual cycles have varying lengths. Coincidence of auricular and ventricular contractions, the former originating in the old pacemaker, the latter in the ventricular pacemaker (idio-ventricular beats), is hardly to be thought of in the presence of this irregularity.

The electro-cardiograms, corresponding to these polygraphic curves, are illustrated by Figs. 12, 14, 15, and 16. The ventricular complexes of Fig. 15 are alone found; the closest comparison between these ventricular complexes and those of the normal periods fails to reveal the slightest trace of auricular representatives buried in the former. The comparison may be made most satisfactorily in the first two cycles of Fig. 14, in which the last nodal beat of a series is shown. Yet we know from the polygraphic curves that auricular and ventricular beats started simultaneously. The conclusion seems justified that the auricular representative is of abnormal form and that it is concealed by R.

A comparison of these electro-cardiograms and experimental ones confirms these explanations.² Auricular representatives may be present, but may be so concealed that the electro-cardiograms require a very close comparison with simultaneous myocardiograms before these representatives are revealed.

² Unpublished observations.

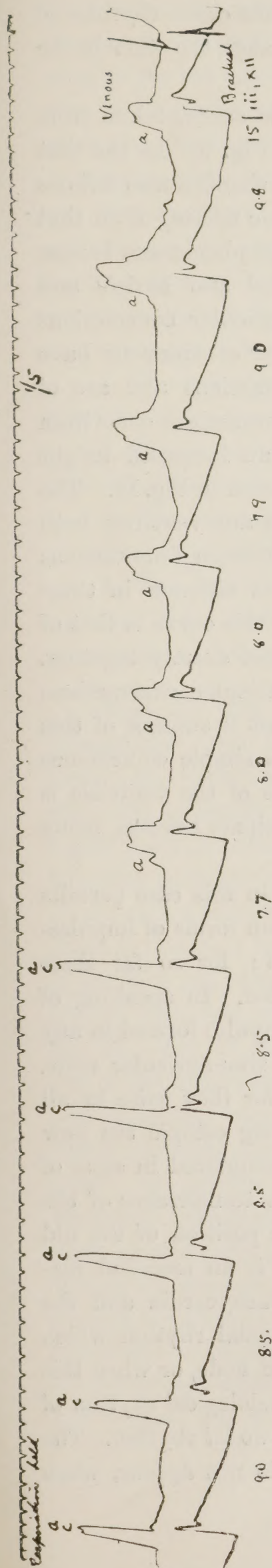


Fig. 3. Polygraphic curve from the same case as Fig. 1, showing the end of the period of slow nodal rhythm and the re-establishment of a slightly irregular sino-auricular rhythm.

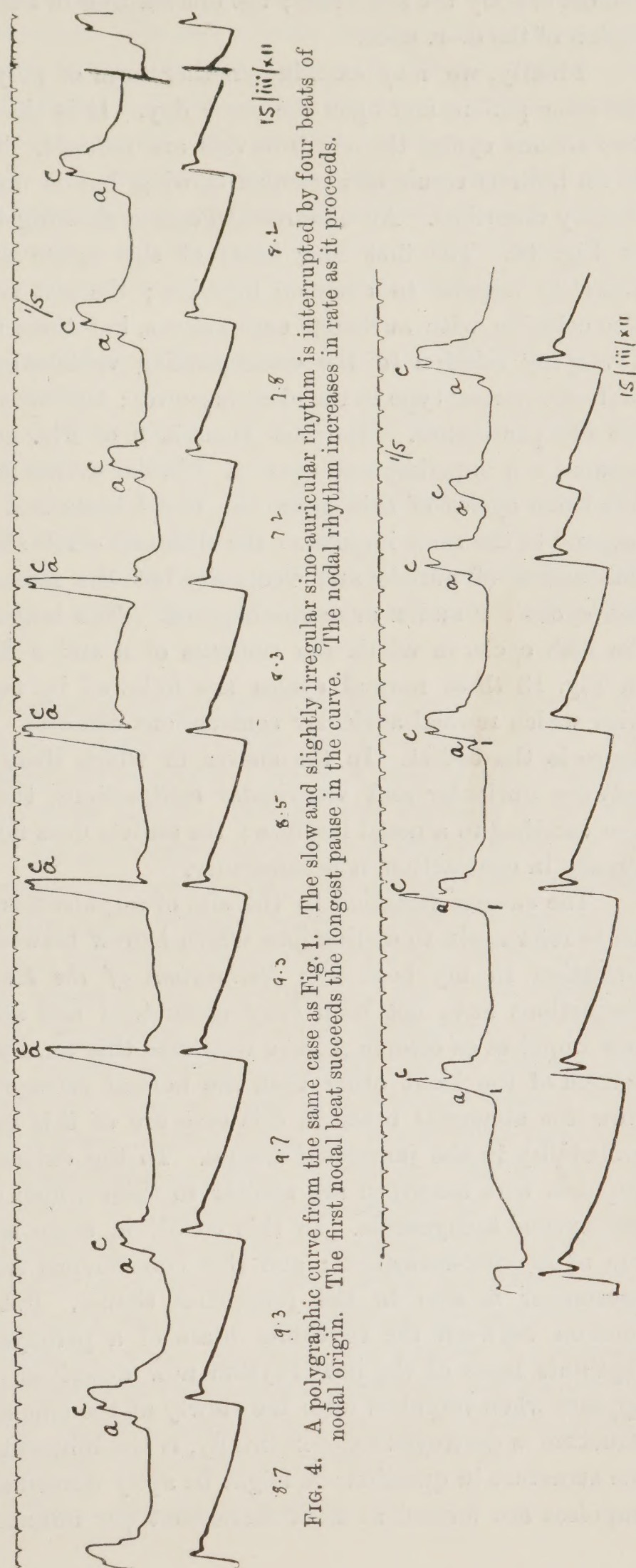


FIG. 4. A polygraphic curve from the same case as Fig. 1. The slow and slightly irregular sino-auricular rhythm is interrupted by four beats of nodal origin. The first nodal beat succeeds the longest pause in the curve. The nodal rhythm increases in rate as it proceeds.

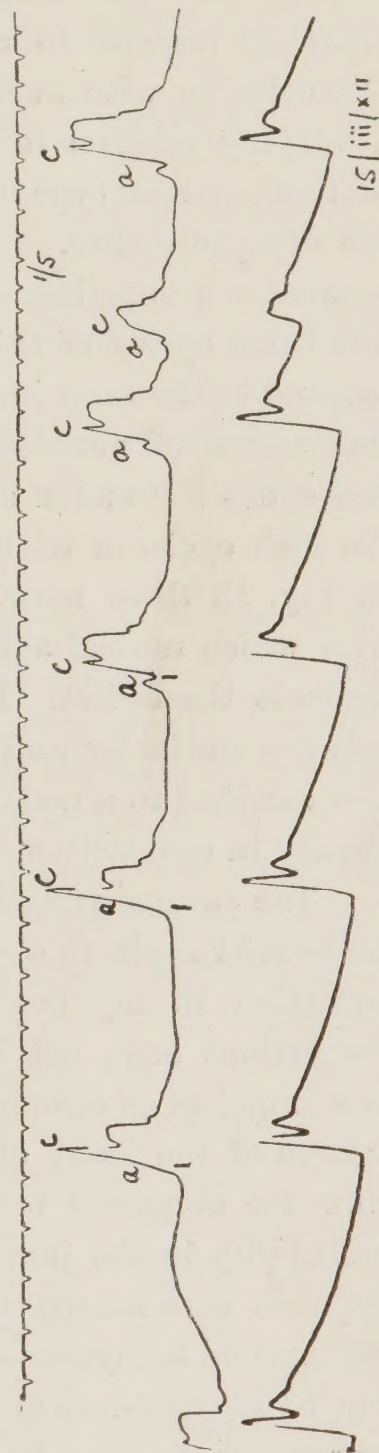


Fig. 5. A polygraphic curve from the same case as Fig. 1, showing two escaped contractions interrupting a slow and irregular sinus rhythm.

Thus we have evidence of two slow rhythms in the same case—rhythms of almost exactly the same rate; the one starting in the pacemaker, the other in the region of the *a.-v.* node.

Finally, we may examine another form of polygraphic curve, taken from the same patient and upon the same day. It is shown in Fig. 5. In the first two venous cycles the *a-c* intervals are reduced. This mechanism also follows as an indirect result of auricular slowing, but is much more fleeting than that already described. An electro-cardiogram showing the same phenomena is seen in Fig. 16. The first four beats of this figure are nodal and auricle and ventricle respond to common impulses; the last two ventricular contractions also coincide with auricular contractions, but these auricular contractions have a varying relation to the corresponding ventricular contractions and are of perfectly normal type in the electric curves; the auricular contractions come from the old pacemaker. The last summit R of Fig. 16 has an increased height because P is superimposed upon it. Similar events may be seen in Fig. 12. The first three cycles of this figure are nodal beats and auricle and ventricle both respond to the same impulses; the fifth and sixth also result from simultaneous contraction of auricle and ventricle, but the mechanism is different in these last cycles; P and R are superimposed. The largest R in this curve is that of the fifth cycle, in which the summits of R and P fall almost exactly together. In Fig. 13 three normal cycles are followed by two ventricular contractions with which normal auricular contractions coincide. The last R summit of this figure is the tallest. In the curves, in which there is this simple coincidence between auricular and ventricular contractions, the escape of the ventricle is also ascribed to a nodal impulse; the auricle does not participate because, being already in contraction, it is refractory.

The curious variation in the site of impulse-formation in this case permits me to refer again to distinctions which I drew between certain forms of impulse-formation in my book *The Mechanism of the Heart-beat*; for so far these distinctions have not been fully understood and appreciated. In speaking of new impulses as ectopic, I have restricted this term to an impulse formed in any portion of the heart other than the normal pacemaker or sino-auricular node. Now the abnormal beats in this case are of this nature, for they arise in all probability in the junctional tissues. To the extent of being ectopic the new impulses here described are similar to those which are encountered in cases of paroxysmal tachycardia. In this condition there is also a transference of the site of impulse-formation; and this transference from the position of the old pacemaker is also to the junctional tissues. But there is an essential distinction between the rhythmic beats of a paroxysm of tachycardia and the rhythmic beats of the new rhythm now described. The nodal rhythm which appears when impulses form too slowly at the sino-auricular node, or when this structure is destroyed experimentally, is the inherent or physiological rhythm of the structure in question; it might be aptly termed an *idio-nodal* rhythm. The impulses are formed at 35 or thereabout per minute and do not appear, when

the heart beats normally, because the old pacemaker is more active and consequently dominates the whole heart. Nevertheless, they are ready to appear so soon as the old rhythm is checked. To such a nodal rhythm I apply the adjective *homogenetic*, and class it under this term both with the old rhythm and with the idio-ventricular rhythm, for they all have the same quality. Homogenetic impulses, as I term them, are not defined according to site of origin. I use the term to distinguish such impulses from those which have the extra-systolic quality, namely, prematurity or rapidity of production; and these, in contradistinction to homogenetic, I call *heterogenetic*. The double classification, including site of formation and quality of impulse, clearly emphasizes the four distinct types of heart-beat.

1. The homogenetic type, arising in the old pacemaker; of which the normal heart-beats are the sole examples.

2. The homogenetic and ectopic type; of which the idio-ventricular and idio-nodal beats are characteristic examples.

3. The heterogenetic type, arising in the old pacemaker; of which the so-called sinus extra-systole is the sole example.

4. The heterogenetic and ectopic type; into which category all extra-systoles, other than those of sino-auricular origin, and all hitherto described paroxysms of tachycardia fall.

Case II. The second series of records which I propose to discuss at the present time were taken from a young man, who was sent to me by Dr. Mackenzie for electro-cardiographic examination. He was usually the subject of slow and generally irregular heart action. Through the kindness of Dr. Mackenzie I am in possession of the polygraphic curve. It is shown in Fig. 6. An irregular pulse curve is accompanied by a venous pulse tracing of the ventricular form; each arterial beat is accompanied by *c* and *v* waves. There is no pre-systolic wave *a*, though a curious wave (marked?) occurs from time to time, the meaning of which is not clear; it is related to the preceding and not to the succeeding systole, but does not appear constantly. The electro-cardiograms are seen in Fig. 17. Fig. 17 (*I* and *II*) shows a regular ventricular beat of constant type and of the supraventricular form. There is no trace of auricular summit in any lead or during any part of diastole, neither are there any of those oscillations which are now known to characterize auricular fibrillation. So far as I am aware, no current interpretation is sufficient in explanation of these curves. Auricular fibrillation is excluded by the regular ventricular action present in these electro-cardiograms and in the left-hand portion of the polygraphic curve, and by the absence of oscillations; weakness of the auricular contraction³ might explain the absence of *a* waves but would not explain the absence of P summits. Simultaneous auricular and ventricular contraction is improbable, though perhaps not definitely excluded, by the type of venous curve. The type of curve (electric and venous) was unchanged when irregularity occurred (Fig. 17, *III*).

³ Venous stasis was not present.

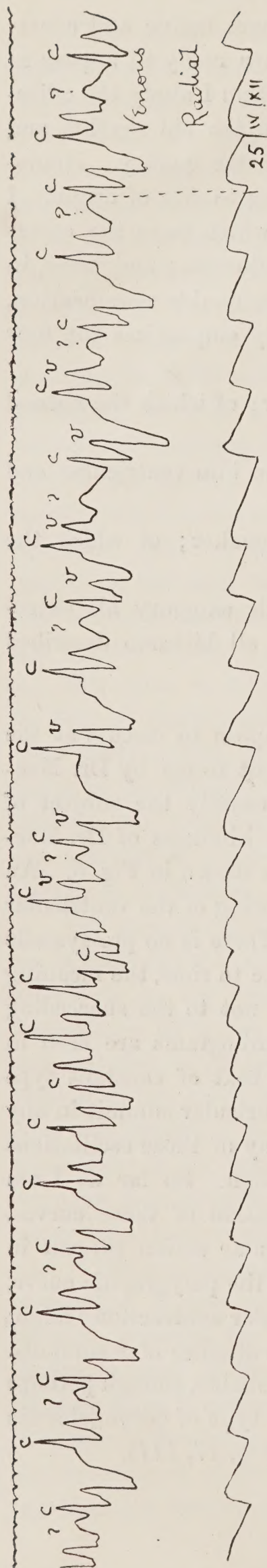
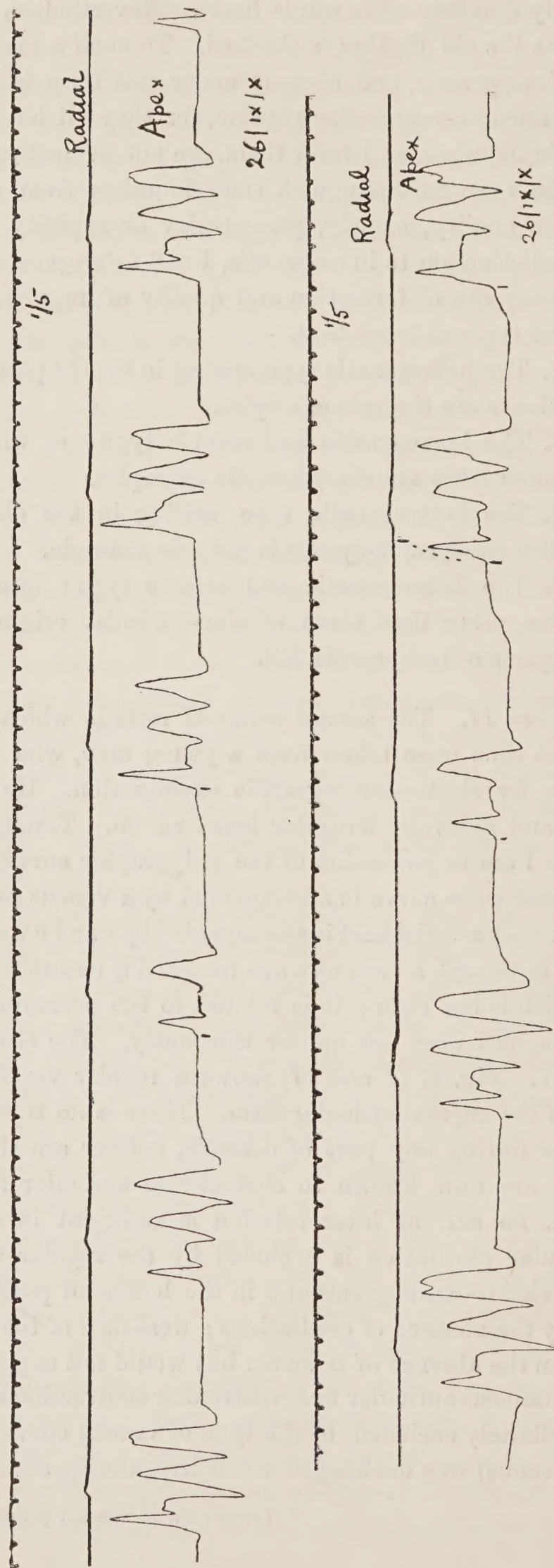


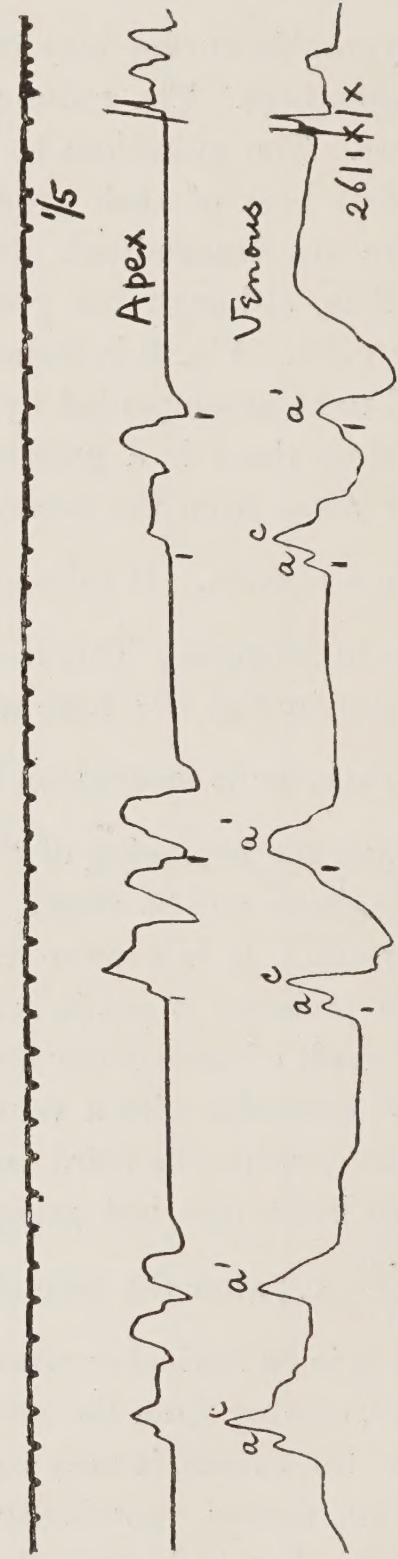
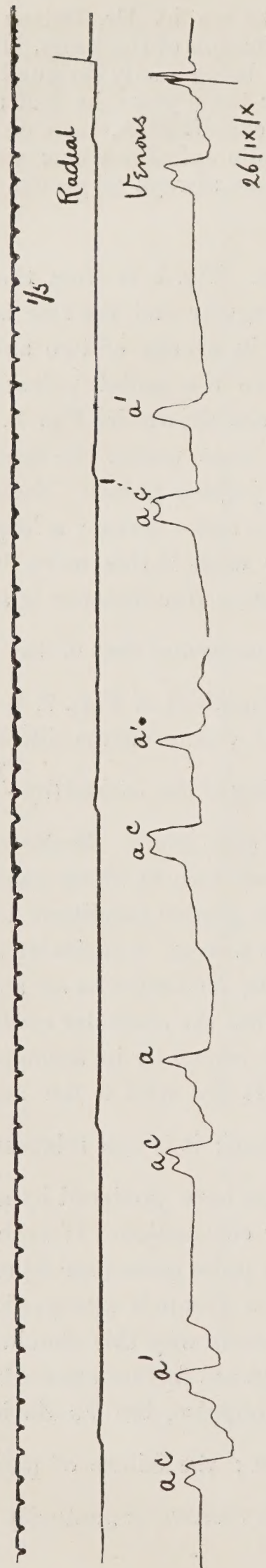
FIG. 6. A polygraphic curve, showing an irregular heart action, accompanied by the ventricular form of venous pulse.



FIGS. 7 and 8. Apex and radial curves. The radial pulse is regular, its rate being 24 per minute. The apex beats occur in twos and threes. The pauses in the radial curve are equal.

Case III. A farmer of 52 years was sent to me by Dr. Batten of the National Hospital in September, 1910, for an examination of the heart. He gave no history of previous illness. He complained of being easily fatigued and of having some shortness of breath on exertion. For three years he had suffered from fits in which consciousness was lost. On examination, increase of the cardiac dullness a half-inch to the left and a systolic apical murmur were discovered. The pulse was regular; the ventricle beat always in groups of two or three.

The polygraphic curves may be described first. Fig. 7 is from the radial artery and apex beat. The radial pulse is quite regular and the rate is about 34 per minute. The pulsations at the apex occur in groups of two and three beats; the first beat of each group corresponds to the radial pulsation, the remainder are not transmitted. Similar events are shown in Fig. 8. It is remarkable that, although the grouping of apex beats varies, the spacing of the groups is even. Fig. 9 is from the radial and jugular pulses. Each radial beat is found to be accompanied by perfectly clear *a* and *c* waves; a long pause follows and then there is a prominent wave. To what is this wave due? It occurs too far away from the commencement of systole (the distance is actually $\frac{3.5}{5}$ sec.) to be a *v* wave. It falls at a time when the second beat of the ventricular group is in progress. This is clear from a comparison of Figs. 7, 8, and 9. It is still clearer in Fig. 10; here we have apex and venous curves side by side. It is not a *c'* wave, for it occurs later than the beginning of the second (by $\frac{1.5}{5}$ sec.) and earlier than the beginning of the third systole of a group. Moreover, it is constant in position and is single, whether there are two or three ventricular beats in the group. It is a wave which is of much greater amplitude than the *c* wave which precedes it, yet it is accompanied by a weak ventricular systole. It is not the result of ventricular systole and must be attributed to an auricular systole, which coincides with a ventricular one. That the auricular contraction has no association with the third ventricular beat is shown by its accompanying groups of two beats (the last group of Fig. 10). It lies with a fair degree of constancy ($\frac{1.5}{5}$ sec.) after the second beat of the group; it is this relation which suggests that it is an auricular contraction which has been produced by a retrograde impulse, coming from the second ventricular contraction. If such is the true nature of the events, it may be asked why the pulse pauses are equal; the reply is that the second ventricular contraction of a group is retrograde while the third is not. It will be seen that there is no reason why this should not be the case; the first and second beats of a given group are further apart than the second and third. The interval for the retrograde impulse, the *Vs-As* interval of the second beat, is large; it amounts to $\frac{1.5}{5}$ sec.; the failure of passage of a second impulse may therefore be the result of the relative prematurity of the third ventricular beat.



The analysis which I have given may appear an uncertain one, because it is based on the interpretation of the curious additional waves in the venous curves. It is substantiated by the electro-cardiograms. These curves were taken on the preceding day; the rate of the pulse was then somewhat slower, being 26 per minute, but a similar mechanism was present. The ventricular beats occurred in couples or groups of three. An electro-cardiogram showing coupling is seen in Fig. 18. The first beat of each group is preceded by an auricular contraction P, and consists of the usual summits R, S and T. The first beat of each group is a perfectly normal heart cycle, in which the *As-Vs* interval (represented by the *F-R* interval) is 0.13 sec. The second ventricular beat of each cycle is anomalous in type. It is a premature contraction arising in the ventricle. Its outline is a familiar one, it corresponds very closely to the type of contraction obtained when the apex of the left ventricle is stimulated in the dog. But it differs from such beats and from similar beats which arise spontaneously in the human heart in one very essential respect. It will be seen to consist of a sharp downstroke, an upstroke, an almost horizontal portion and finally a slow rise and fall. But it is malformed at one point. The anticipated outline should follow the dotted line on the middle group of the curve. The curve is notched at the beginning of the slow rise. The notch is placed $\frac{3.3}{5}$ sec. after the commencement of the first beat of the group and $\frac{1.2}{5}$ sec. after the commencement of the second beat of the group. The relations of the curious waves in the venous curves (Fig. 9) to these points may be remembered: $\frac{3.5}{5}$ sec. and $\frac{1.5}{5}$ sec., a sufficient approximation, seeing that the curves were taken on different days and by different methods. The notched electro-cardiogram is the result of the same event as the venous wave in question; both are due to an auricular systole which falls with the second ventricular beat of a group. But its electric representative is directed downwards, while the usual auricular representative for this patient is an upwardly directed summit. The auricular contraction is an abnormal one; it has travelled in an abnormal manner through the auricle. Such an auricular electric complex is anticipated when the contraction commences below and travels upwards, as it would do were the auricular contraction propagated from the ventricle. The electro-cardiograms of the coupled periods consequently confirm the analysis of the venous curves.

A single consideration remains. If the first premature beat is retrograde and the second is not, then the electro-cardiograms of the groups of three beats should be of material assistance. The second and third beats of the group might be compared and it might be expected that they would differ only in regard to the notching, the second beat of the group being complicated by an abnormal auricular contraction, and the third beat of the group being free of it. The actual comparison however is not so easily accomplished, for the reason that in none of the curves are the second and third beats of a group identical. Some-

times they are totally different, and in all there is some divergence. Nevertheless the curves are sufficiently clear. Fig. 19 is an example. In the first group of this figure there are three ventricular cycles: the first is normal in every respect; the second is of the type seen in Fig. 5 and is distinctly notched; the third is of a new type, and the point at which the notch is expected is marked on the curve; no such notch occurs. The second group is a clearer example, for the two premature beats are alike in general form. The first of these premature beats (the second of the group) is notched, the second premature beat (the third of the group) is not distorted in the same fashion.

To sum up, the electro-cardiograms furnish corroborative evidence that the analysis of the venous curves, as it has been given, is correct, and that we are dealing with retrograde extra-systoles.

Conclusions.

Three cases of slow heart action of unusual form are described.

Case I. In a man who presented a continuous slow action of the whole heart, irregularity of the ventricle was observed. This was due in part to the building of homogenetic impulses at two centres and at almost equal rates. The centres of impulse-formation lay at the normal pacemaker and in the region of the *a.-v.* node.

Case II. Curves are described from a young subject who displayed slow and at times irregular heart action. The disorder, as illustrated by polygraphic and electro-cardiographic curves, was of a hitherto undescribed form. No trace of auricular contraction, either co-ordinate or inco-ordinate, could be discovered in any record, venous or electro-cardiographic.

Case III. A case in which the Adams-Stokes' symptom-complex was present is described. The pulse-rate was from 26 to 33 beats per minute. The ventricle beat in grouped fashion; the first beat of each group was of normal origin; the second beat of each group was premature and arose in the ventricle, it was also retrograde; the third beat, when it occurred, was premature and arose in the ventricle, but was not retrograde, and the pulse was consequently regular.

EXPLANATION OF FIGURES.

PLATE 14, FIG. 11. Electro-cardiograms from Case I, showing the three leads, *I*, right arm to left arm; *II*, right arm to left leg; and *III*, left arm to left leg. In leads *I* and *III* the heart is beating regularly at a rate of 54 per minute. In lead *II* the heart is beating irregularly, but the beats are of the same nature, being generated in the sino-auricular node. The horizontal lines in this and subsequent figures divide spaces equivalent to 10^{-4} volts. The time-marker is in $\frac{1}{30}$ seconds.

FIGS. 12-16. A series of curves from lead *II* from the same case; the lengths of the cycles are marked in thirtieths of seconds. Figs. 12 and 16 show three cycles in which auricle and ventricle respond to common and nodal impulses and two escaped contractions in which the ventricular beats are coincident with normal auricular contractions. Fig. 13. Three normal cycles are followed by two escaped contractions. Fig. 14 shows a single nodal beat followed by four normal cycles. Fig. 15 shows five nodal beats.

PLATE 15, FIG. 17. Electro-cardiograms from the three leads in Case II. In leads *I* and *II* the action of the heart is regular, the rate being 44 per minute. In lead *III* the heart action is irregular. In all the curves the mechanism is similar; the ventricular beats are of a supraventricular type and there is no sign either of a P summit or of the oscillations which characterize a fibrillating auricle.

FIG. 18. An electro-cardiogram from Case III (lead *II*), showing a bigeminal action of the ventricle. The first cycle of each couple consists of a normal sequential beat, the second beat of each couple is a premature beat, presumably arising from the left side of the heart. It differs from the beats known to originate in this neighbourhood in that it is notched. An inverted auricular complex follows with this anomalous ventricular complex and distorts it, as shown by the dotted line in the middle group of the figure. The figure illustrates retrograde heart-beats. The time-marker in this and Fig. 19 is in $\frac{1}{5}$ sec.

FIG. 19. An electro-cardiogram from the same case (lead *II*), showing the heart-beats grouped in threes. The first and second beats of each group are similar to those of Fig. 18. The third beat of each group is also of ventricular origin but comes from different points in the two instances. No trace of auricular contraction can be found to complicate this third beat. A comparison may be made between the two premature ventricular contractions in the right-hand group; the first of these is distorted by an inverted P, the second is not.

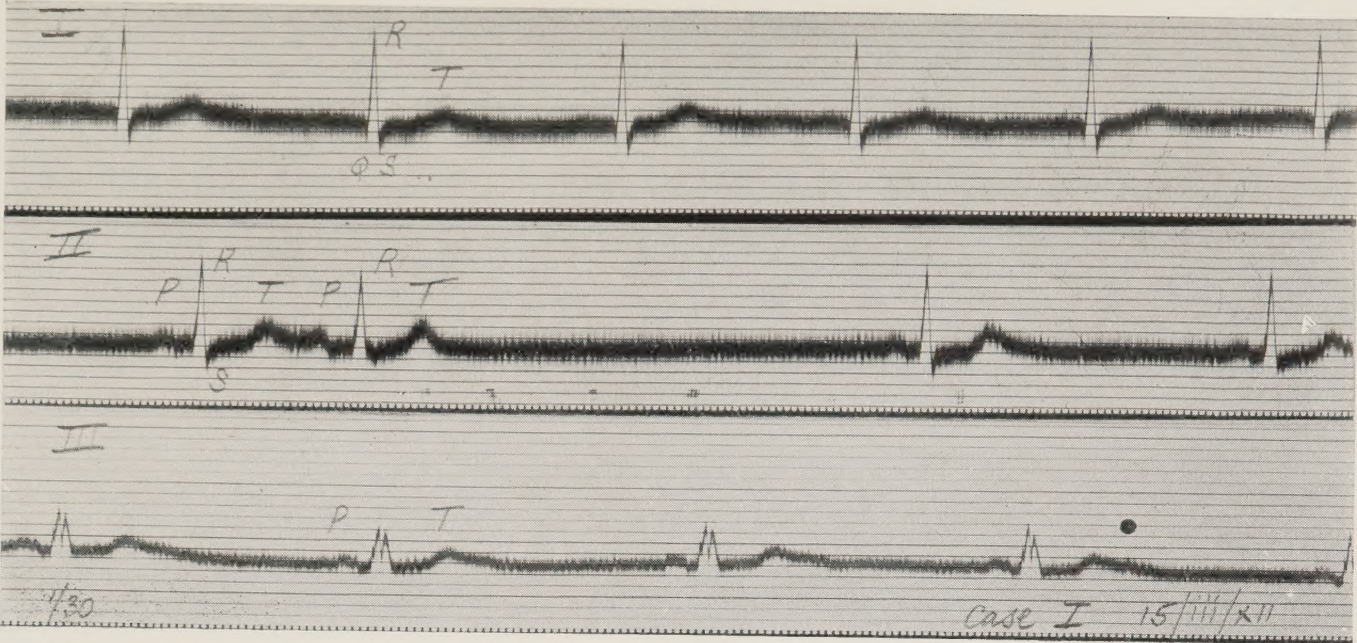


FIG. 11

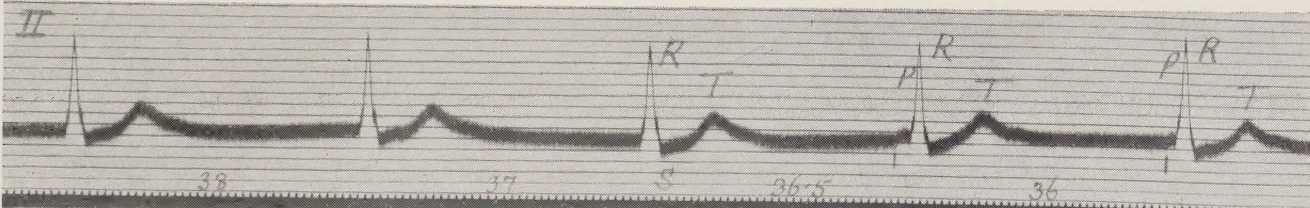


FIG. 12

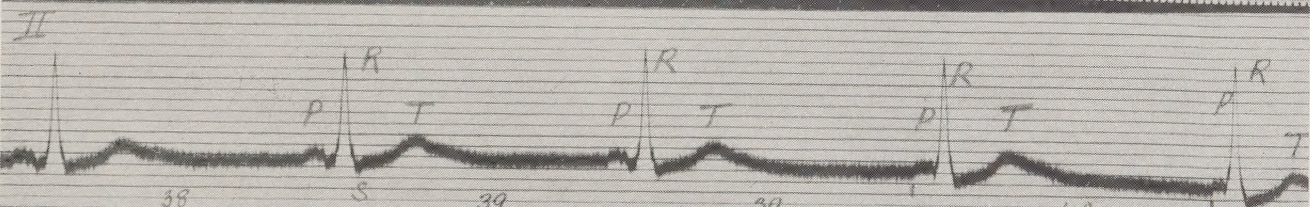


FIG. 13

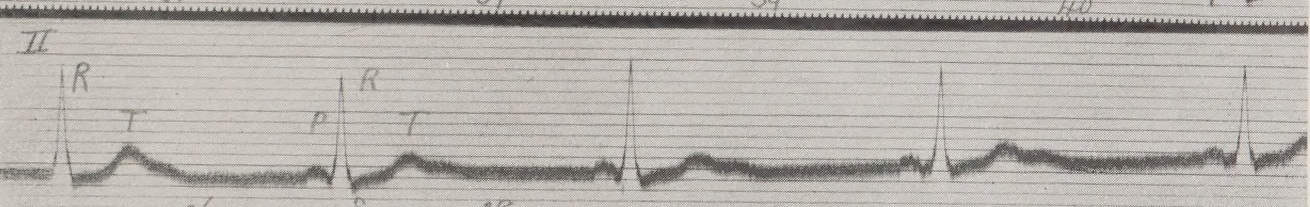


FIG. 14

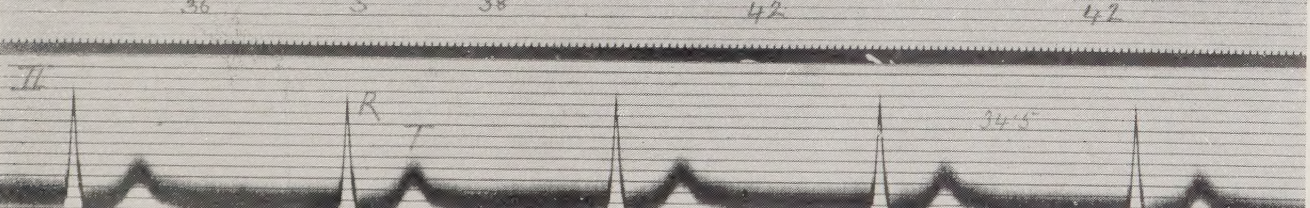


FIG. 15

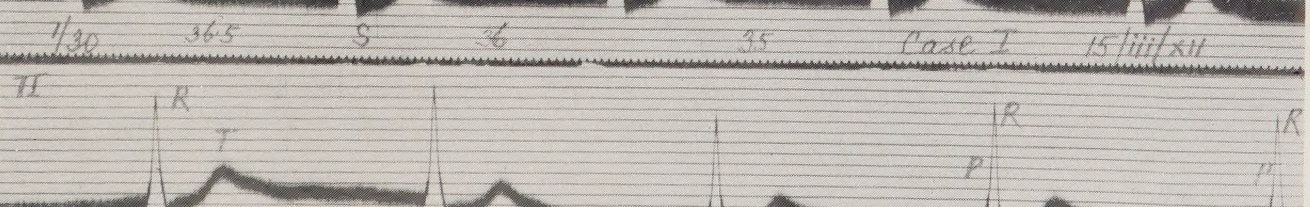


FIG. 16

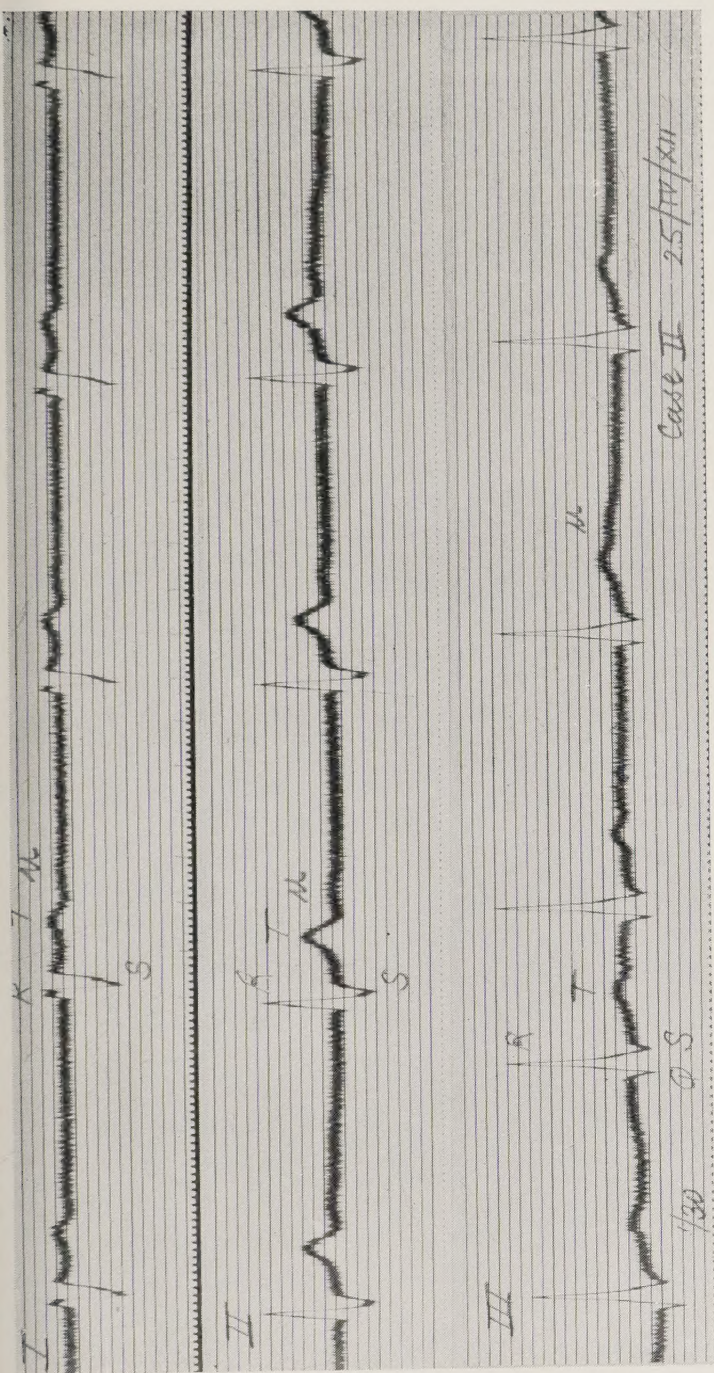


FIG. 17

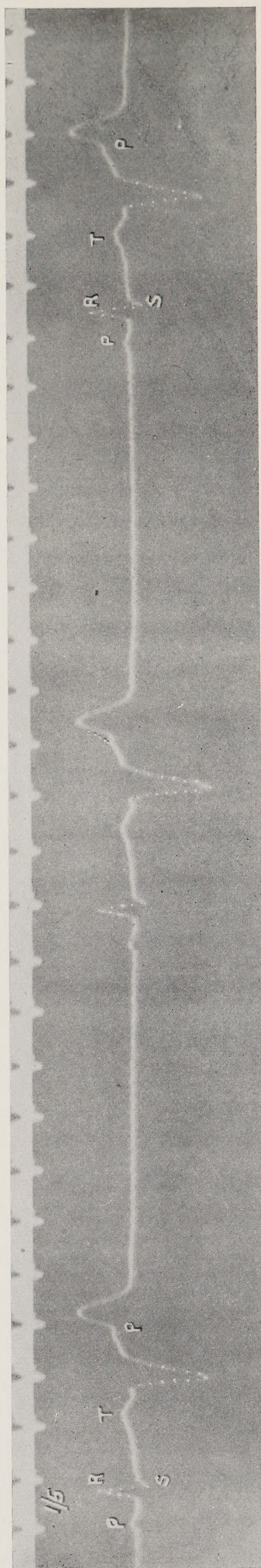


FIG. 18

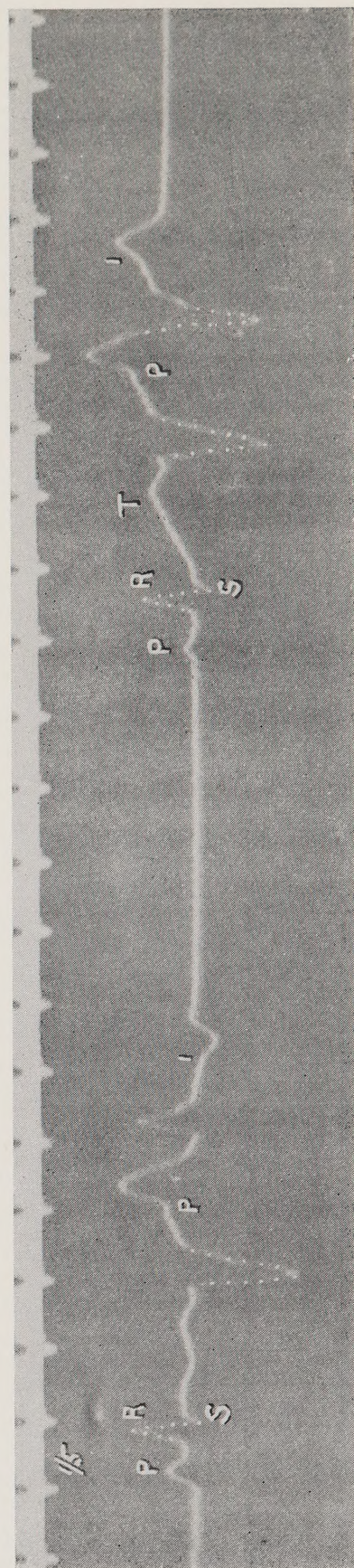


FIG. 19

